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SOCIAL STRUCTURES AND MENTAL DISORDERS: COMPETING HYPOTHESES OF EXPLANATION

H. WARREN DUNHAM, PH.D.

THE central focus in this paper will be to examine critically the kinds of hypotheses that have been advanced to explain the findings of some epidemiological and ecological studies of mental disease. In order to accomplish this task it is necessary to point to: (1) the differences between epidemiological and ecological studies; (2) the various theoretical considerations directing these studies; and (3) the value and purpose of both kinds of studies in understanding some of the problems associated with mental health and disease.

As I have reflected on this problem it seemed to me that some clarification of the task will result by pointing to the distinguishing features of epidemiological and ecological studies of mental illness.[1] Epidemiology has been defined as "the sum of what is known regarding epidemics." (8) As such, one most important phase of an epidemiological study is to find out the incidence and prevalence of a disease in a circumscribed community setting or a carefully delimited population group. The general objective in these studies has been primarily an overall count of cases. Furthermore, most of the epidemiological studies, particularly the European ones, have been conducted by medically trained persons who have regarded their efforts as having some genetic significance. Thus, when they compare the results of their surveys they think that their theoretical position is supported when the statistical differences are at a minimum. For example, the two well-known surveys in this country, the Baltimore (6A, B) and the Williamson County, Tennessee, (39), reported rates for active and inactive cases of 62.0 and 69.4 per 1,000 population respectively. Such results frequently have been interpreted that regardless of the type of area—

[1] While in the work of epidemiologists and ecologists there is much overlapping of interests and technique, I have elaborated this distinction, even though it may not be acceptable to some, with the express purpose of sharpening up existing disagreements and expressed viewpoints.

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whether urban-north or rural-south—the frequency of mental and behavioral disturbances in a delimited population is about the same.

However, epidemiological comparisons in the hands of social scientists may produce a different emphasis. Thus, Eaton and Weil (9a) using a standard expectancy method, compared ten surveys relative to the amount of enumerated psychoses. The Hutterites, serving as a norm, had a higher enumerated rate of psychoses than seven of the ten populations upon which they reported. They regard the high Hutterite expectancy ratio as a function of their thoroughness in screening and think that five of the populations ranking below the Hutterites were not screened thoroughly for their recovered mental cases. After several other arguments they conclude that the Hutterites have the lowest frequency of psychoses among the four rural populations but higher than the frequency found by Tsung-yi Lin in his survey of Formosa (28). They then point to the limitations of the epidemiological method with its capacity for showing the presence or absence of quantitative differences but without being able to explain them. "Their genetic composition, physical health, psychological tendencies would have to be analyzed in detail with the hope of identifying some patterned interrelationships that could account for the quantitative differences." (9b)

Even so, undeterred the investigators proceed to show that a sociological orientation is useful in accounting for the predominance of the manic-depressive psychosis (73.6 per cent of all persons diagnosed as psychotic among the Hutterites). After convincing themselves that their diagnostic judgments were valid, they develop the role of the "social cohesion factor." From examining various bits of evidence they conclude that the ratio of manic-depressives to schizophrenics varied in different population groups and this fact (they call it an assumption)"—would support the general theory that sociological factors play an important role in the way functional mental disorders are manifested in patients." (9c) They conclude tentatively, "The

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extreme cohesiveness of the Hutterites which may contribute to their low frequency of schizophrenia, may be significant for the relatively high proportion of manic-depressive reaction among psychotic members of the sect." (9d)

I have used Eaton and Weil to illustrate the fashion in which the orientation of the investigators enters into an interpretation and also to illustrate the gap between a theory and the evidence to support it. (Weil, of course, is a psychiatrist.) That they should fasten upon the differential frequency of these two functional diagnoses among the Hutterites to show the relevance of sociological factors is incredibly naive considering the seemingly interminable problems in making the differential diagnoses and considering also that many different psychiatrists were involved in making the differential diagnoses in the other studies.

It might have proven just as valuable to make something of the fact that Bremer's Arctic Norwegian village (1a) had a frequency of psychoses that was twice the Hutterite rate, for in both studies the screening was supposed to be excellent and the data were gathered in extremely isolated communities. If we assume this difference to be a real one, then one is forced to conclude that the population of the Norwegian village is genetically inferior to the Hutterites or that the conditions of life provide a more telling impact upon the people of the Norwegian village than a different set of life conditions do in the case of the Hutterites. And this in a final sense is the big factual question—"Can such surveys as counts of prevalence and incidence of total psychopathy or particular disorders show significant quantitative variations between different cultural groups and/or different strata or categories of population within the same society?"

Now, let us turn to consider the ecological studies of mental illness. Ecology has been defined as "the science of organisms as affected by the factors of their environment" or "the study of the environment and life history of organisms." (8) This means that an investigator who wants to make an ecological

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study of mental disease will be concerned in locating the mentally diseased person in some position within a culture or social system and viewing him in relation to the positions occupied by other mentally diseased persons and the non-mentally diseased in that system. Thus, an investigator making an ecological study of mental disease, or a mental disease, is concerned with discovering if the incidence of disease, or a given disease, will vary significantly between different temporal, spatial or social environments. These environments are frequently delimited as social classes, religious groups, occupations, types of families, types of communities, levels of education, historical time, or special environments such as school, military unit, or prison.

Now, if the ecologist can show conclusively that certain significant rate differentials for mental disease exist between different positions in one of these environments, he faces the task of trying to find some hypothesis derived from a theory that may explain the difference. Thus, most of these studies have been largely empirical in character and if any theory is pertinent, it is of the broadest and most general variety.

However, the ecologist focusing on the organism in his environment will be concerned with trying to isolate certain environmental factors that will explain the rate differences and that will account for the development of the disease in the person. If he operates as an ecologist he will emphasize the processes within the environment and attempt to show the social variable or complex of variables that is associated with the rate differential. If he tries to get at the social factors that are causative or predisposing for persons in that environment, he will be thrown on another level of analysis where his ecological findings will prove only indicative of some factors that he might study as having an etiological significance.

The difficulties and pitfalls that beset the investigator in this area as he strives to make sense out of his findings are well illustrated by Frumkin's study of occupations and mental disorder (13a). He begins by emphasizing that research is needed to investigate the etiology of mental illness. His data comprised

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all first admissions to Ohio State's prolonged-care mental hospitals for the year 1950, who had an occupation, and who had not been classified as housewife, student, or unknown occupation prior to admission. These data (1,192 males and 347 females) were then broken down by occupation in relation to age, sex, and mental disorder. His basic findings consist of rates per 100,000 for each sex for the major psychoses in twelve general occupational categories ranging from unskilled to professional.

It almost goes without saying that his data, method, and findings raise many questions that should be considered before attempting some theoretical interpretation. The finding that the low prestige occupations have the highest rates of first admissions for the major mental disorders is—all things considered—hardly startling. However, without even touching on the difficulties here Frumkin barges joyously ahead and using the "method of *Verstehen* rather than by strictly empirical methods" proceeds to all kinds of generalizations about man in American society which are not only dubious in general but also in relation to his findings which should be boxed in by many qualifications. To illustrate with a few choice quotations:

> A man's occupation, in general, is more imporant to his mental health than is the occupation of a woman to her mental health.
>
> Thus, we find that the more radical male and female unskilled and service workers have the highest rates of alcoholic and syphilitic psychoses because, next to criminal acts, sexual promiscuity and alcoholism seem to be the best emotional outlets, the best known escape from hostility, the rejection and general frustation which are so often encountered among people in the lower socio-economic strata of our society.
>
> To go a step further, one might say that the etiology of lower socio-economic-status-group mental illness (and crime) is generally socio-genic in nature, whereas in the upper strata of society, mental illness is generally more psycho-genic. Thus, the etiology of middle class mental disorders, being somewhere in between, i.e., more or less equally socio-genic and psycho-

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genic, or simply psycho-genic in origin, reflects characteristics of both extreme strata of society (13b).

I have used Frumkin's study here not because he reported his findings, but because his study illustrates well the trap of excessive sociological zeal as one moves from rate differentials to some theoretical interpretation. His quality of interpretation is too strained, but hardly drops as the gentle rain from heaven.

Turning now to the general theories that have directed both types of studies, I have already indicated that they are of the most broad and general kind. The epidemiological studies, particularly the European, have stemmed from a broad biological basis that attempts to point up differences or similarities which must be inferred about the genetic composition of a given population. Investigators with this orientation are likely to be more intrigued if the rates in a given population group or between different population groups are approximately equal.

The ecological studies, largely American, by contrast are likely to be guided by some broad sociological theory which attempts to show how certain social factors may be psychotic-inducing for persons occupying a given position in time or social space in a social system. From this broad theoretical basis excitement is high when a distribution of cases in relation to some aspects of social structure shows that the rates vary significantly from the different positions in that structure. The question, then, immediately arises: How certain can we be that our findings actually depict a "true" rate difference in the incidence of the disease or diseases under study? This is the rub, without doubt, for the fact is that with respect to both types of studies controversy has centered around the problem of validity. In general, those investigators with a bio-organic orientation try to show that significant rate differences of incidence in a social structure are not valid and cannot be accepted, while investigators with more of a sociological orientation attempt to show that they do possess a validity. Then there are, of course, the

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stolid empiricists who attempt to examine the evidence with an impartial and objective eye.

I wish now to comment briefly on the purpose and value of these studies for an understanding of mental health and disease. It seems to me that these studies, considered jointly, have five central purposes and can be valued to the extent that they prove useful as aids to detection, diagnosis, treatment, and prevention of mental disorders in human society. First, these studies frequently serve the purpose of administration and can be helpful in pointing to the quantity and quality of services and facilities that may be needed in the future. Secondly, they may serve the purpose of sharpening our devices for detecting and screening the mentally ill in any population group. Thirdly, they can provide us with some conception of the size and extent of mental disease in general, or of a specific disease as it may exist in a given population group. Fourthly, these studies can add to our knowledge of social systems by showing those elements of processes within such systems that are associated with high and low rates of mental disturbances. And finally, they can sometimes be suggestive of hypotheses bearing on the etiology of mental disease that must be investigated by other methods and that must serve as a testing ground for the crucial relevance between biological and social psychological theories.

With these rather lengthy introductory remarks, I return now to my central task which is to critically examine the hypotheses utilized to explain findings of selected epidemiological and ecological studies of mental disease. This task has three parts. First, I wish to analyze briefly the central problem with which this type of research is always confronted. Secondly, I want to point to some of the characteristics of our data that present much difficulty for this survey-type of research in mental illness, especially in the complex, technologically advanced societies of the West. And finally, I intend to examine the hypotheses that have been utilized to explain rate-similarities or differences in various types of social structures. I further intend to examine these hypotheses as they have evolved in the

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numerous studies that purport to find the incidence and prevalence of mental disease in different positions of the social structure or in specific delimited population groups. However, I do not intend here to cover the total literature, which is voluminous, but to confine my citations and illustrations to what, in my judgment are some of the most significant studies that have appeared during the last two decades.

The central problem which these studies present is posed by the question: "How does one define or delimit the case?" Now, this has been examined in other contexts (30) and I do not wish to belabor it here. But the problem continues to be crucial and is generally brought up when the results of such studies are presented. Currently, there are three devices: (1) There is the arbitary definition. Thus, we will count all persons who seek help from psychiatrists, clinics, or hospitals for the first time, all persons entering a mental hospital for the first time, or all persons being treated at a given time. However, if we arbitrarily define the case, how do we know if coverage was complete enough to assert the reliability of the rates as found so that any interpretations of them will have some validity?

(2) One can argue, as I have on occasion, that who is counted as sick in any social milieu is the result of a social judgment made by family members, friends, or neighbors and a judgment that the person must accept in some fashion by taking action or having some action taken upon him by others. This means that judgments about who is mentally disturbed or can be regarded as a mental case will vary in different social milieus, communities, and sub-cultures. This definition stacks the cards and contains within it a built-in explanation for rate variations at the different positions in social structure. This position also implies that the way to reduce mental disturbance in a high rate group would be merely to conduct some kind of educational campaign to bring about greater acceptance and toleration of human frailties. If this position has any relevance it probably would be to the minor type of disturbances, but this then becomes a clinical problem. How does the diagnostician deter-

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mine that he deals with a minor personality distortion and one that is not a forerunner of a more severe disturbance?

(3) Of course, there has always been the hope that for those types of mental illnesses where etiology is still obscure, biochemical research will eventually find the answer and in so doing will perfect a test so that we can say with complete assurance that a given person has a given mental disease. Some clinicans have felt that these statistical-survey-type studies are useless without such an objective test. However, one might say in their defense that while they have contributed little of etiological significance, they have helped to clarify certain issues in the mental health field, to be revealing of our social systems and to point up certain hypotheses that might be investigated by other methods.

In addition to the problem of defining the case, there are several other characteristics of our data that make the conducting of this type of research quite difficult, especially with respect to our confidence concerning reliability and validity. Here, I have reference to the following factors: the great mobility of patients that is a reflection of stepped-up mobility in the entire society during the past half-century; the discrepancy between the onset of mental disease and the decision to seek treatment; the great spread of age at which patients are first recorded as having a mental illness, and the great variety of diagnostic types with numerous remissions in the various categories over the years. In addition, there is also the great difference in policies and administrative practices of states and countries that make it difficult to secure comparable data. Finally there are always the great variety of theoretical orientations that exist among psychiatrists as they attempt to arrive at a diagnostic judgment.

Now, the central issue in all these studies is whether there are reliable and valid rate differences for mental disease, or for a specific mental disease, between different positions in a social structure or a sub-culture. Consequently, in the remainder of this paper I will be concerned with examining the hypotheses

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that have been advanced to explain rate variations among the different positions within a social system and also to show the manner in which various studies have tended to classify them or advance them as interpretations of their own findings.[2] Thus, I am not concerned in this analysis with pointing to the various correlational findings of mental disease rates and different indices for status, isolation, or population characteristic that have appeared in the literature. For the function of these correlations is merely to add support to rate differentials that a particular investigator has found present in a particular social structure. That schizophrenic rates are high in low income occupations and in occupations of low prestige as Clark has reported (4) or that the rate of mental disease in certain subregions of Texas shows a correlation with the number of psychiatrists in private practice of .40 as Jaco has reported (21) will not concern us here. For such correlations, quite numerous in the literature, while interesting, have added nothing with respect to throwing any light on the social etiology of mental disease or a specific mental disease.

The crucial problems concerning rate differentials are three in number. (1) With what confidence can we assert the reliability and validity of rate differentials or no rate differentials for mental disease as found in specific social structures? (2) What is the most plausible interpretation of the presence or absense of such rate differentials? (3) Does a particular interpretation provide us with some suggestive hypotheses about social causation that we can subject to some crucial test?

There are two classes of hypothetical interpretations concerned with providing adequate explanations for rate differentials of mental disease in social structures. These are (1) the

[2] While in accordance with my assignment, I have confined my analysis to those studies dealing with position in the social system, I am constrained to point out that the same problem arises when making cross-cultural comparisons. For example, consider the following questions: Does a given culture have more mental disease than another culture? Does a given culture have a higher incidence of one type of mental disease than does another culture? The problem as to whether all cultures have basically the same kinds of mental disease is relevant to a cultural analysis but is not pertinent to our concern.

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non-theoretical hypotheses and (2) the theoretical hypotheses. By non-theoretical hypotheses I refer to those explanations that stem from no acceptable body of scientific theory but find their *raison d'etre* in some defect of method or some obstacle in the environment. Theoretical hypotheses refer to propositions that derive from or can be derived from some acceptable body of scientific theory. Within the class of theoretical hypotheses, we wish to distinguish three sub-classes—those on biological, social psychological and social system levels.

Let us turn to a consideration of four non-theoretical hypotheses. All of these have been suggested at one time or another for the purpose of showing rate differentials that have been reported as incidence rates are in reality not "true" incidence rates. Perhaps the oldest and perhaps the fairest argument has been the notion of "incomplete coverage." From this point of view it is contended that a given investigator has not included in his count all the new cases occurring in an area mainly because he cannot get to them. Thus, if he counts only first admission to mental hospitals, his count is off because he does not include admissions to outpatient clinics and private psychiatrists. If he includes these his count is off because he does not take account of those in the community who are sick but who do not get into treatment. This argument has been faced by numerous investigators in their studies in one way or another including Faris and Dunham, (12a), Goldhamer and Marshall, (15a), and Hollingshead and Redlich (19a). This, of course, is the demand for a "true" incidence figure, and until it can be secured many are likely to remain skeptical of any significant rate differentials that are reported.

In the nineteenth century the one readily acceptable non-theoretical hypothesis was the "law of distance." This "law" merely asserted that the rate of first admissions to a hospital varied inversely with the distance from the hospital. This is equivalent to a contemporary non-theoretical hypothesis that views variations in the first admission rates by geographical or status positions as merely a reflection in the differential avail-

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ability of psychiatric facilities, whether hospitals or beds. This was one of Kramer's (23) conclusions when he studied the admission data from 1916–1950 at the Warren State Hospital in Pennsylvania. Ødegaard (34) reports that rates of admission vary in the different areas of Norway because the more and better facilities tend to account for the higher rates. However, in another study of the incidence of mental disease during World War II, Ødegaard (35) concludes that loss of facilities cannot explain the 1941–1943 decrease in rates. He notes that decreases were particularly marked in Oslo where facilities were fairly good and least marked in the northwest where the bed shortage was especially bad. He also notes that if facilities determined the decrease then there should be a decrease in nursing cases and readmissions but in these series there was an increase. He finally concludes that even though a number of psychoses seemed directly caused by the War, there was a real decrease in psychiatric morbidity and a net gain in mental health. This finding will concern us further when we attempt to assess those theories on the social system level.

Malzberg's (29a) evidence on this point for New York State is also negative. He shows that between 1925 and 1935 bed capacity increased 71.4 per cent while first admissions only increased 58.3 per cent. He thinks that this fact combined with evidence from other states seriously questions the proposition that bed capacity determines the rate of first admissions. Even so, as Ødegaard has pointed out to me an increase of bed capacity at a given point in time is, as a rule, followed by a jump in first admissions the following year.

There is, finally, the statistical criticism. While this stems from an acceptable body of statistical theory, I have called it a non-theoretical hypothesis because it disposes of rate differentials by claiming some defect in the collecting of or handling of the data. Thus, this hypothesis points to inadequate sampling, failure to establish significant rate differences, and inadequate number of cases—thus, by increasing the cases by three or four in any cell the entire picture might change—or a mobile popu-

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lation. Any of these items might destroy any rate difference reported.

I turn now to a consideration of our first set of theoretical hypotheses: those concerned with a biological level of explanation. In general, these studies have been largely of European origin and have been directed to (1) establishing valid estimates of the "true" incidence and prevalence of mental disease and specific mental diseases in various populations, (2) collecting statistical evidence for the genetic linkages of mental diseases through specific family groups and/or inbreeding of populations (1b) and (3) obtaining reliable frequency figures for a population in order to compare them with frequency found in hereditary tainted families.

I do not intend to review all of these European studies but will point to two of them which illustrate the above points. Torsten Sjögren (41a) reported on the investigation of the occurrence of psychoses and oligophrenia during the period January 1, 1900, to December 31, 1944, on an island, A:bo, off the west coast of Sweden. He states, "The object of the investigation was a thorough statistical and hereditary-biological analysis of psychoses and oligophrenia as well as genealogical survey of the cases concerned and their facilities as far back as it was possible with the aid of parish registers and archives. . . . Furthermore, statistical investigations regarding heredity and incidence of mental disorders in the parents and siblings of the probands can be made and the extent of inbreeding analyzed" (41b). His data, after diligently searching the records, included 397 persons of which 335 comprised his proband group and 62 were secondary cases (siblings of probands). Of the total, 397 persons, 158 comprising 117 families were assembled into a connective pedigree complex. His findings broken down by pedigree, parish, birth, and residence, and diagnosis are voluminous, and while impressive, Sjögren presents them with no interpretation other than implied genetic explanations. His overall finding showed a prevalence rate of 11.0 per 1,000 population for psychoses and 5.7 per 1,000 for oligophrenia. He finds

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this in excess of figures for all of Sweden in 1940 which were reported as 4.5 and 2.9 per 1,000, respectively. These latter figures he thinks, are under-enumerated. On the other hand, he finds that his figures are quite close to Stromgren's figures for the island of Bornholm in 1938 reported as 11.4 and 4.2 per 1,000, respectively. This comparison is reminiscent of that reported by Goldhamer and Marshall (15b) when they call attention to the fact that Kurt Fremming's expectancy for figures for various causes of mental disorders up to the age of 56 in the same island of Bornholm (1951) are of the same order and magnitude as those expectancy measures that Goldhamer and Marshall calculated for New York State.

In view of my distinction between the epidemiological and ecological studies of mental cases, it is of some interest to note the contrast between the distribution of Sjögren's cases in his pedigree complex as compared with those cases outside the pedigree complex in four parishes of A:bo. While Sjögren does not present these figures, a slight computation based on the population of the parishes which he has given us, reveals it. My computed rates on Sjögren's figures are shown in the accompanying table.

One notes interestingly enough that the cases in the pedigree complex are more highly concentrated in two of the parishes that are poorest in terms of social economic level while the cases outside the pedigree complex are distributed rather evenly over the four parishes although the higher rates are in the other two

Number and prevalence rates of cases of psychoses and psychopathy in four Parishes of A:bo in and outside of pedigree complex.

Parish	Average Population 1900–1944	No. in Pedigree Complex	Rate[1]	No. Outside Pedigree Complex	Rate[1]	Total Cases	Rate[1]
S	4,071	89	21.9	71	17.4	160	39.3
V	1,749	33	18.9	28	16.0	61	34.9
R	2,334	7	3.0	49	21.0	56	24.0
K	1,232	1	0.8	30	24.4	31	25.2

[1] Rate per 1,000 general population.

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parishes. I am pointing this out because it seems contrary to what might be expected. Here, the expectation would be that the cases inside the pedigree complex showing hereditary linkages should be distributed more evenly over the four parishes, while the cases that supposedly have no genetic linkage should be more heavily concentrated in the two poorest parishes. This may, however, mean that the sick people on this Swedish island who are sick because of some genetic characteristic find themselves in much poorer circumstances than those who are sick for other reasons. Thus, those who are sick for other reasons come largely and somewhat evenly from all the social classes in this particular Swedish island.

Bremer's study (1a) of the frequency of psychiatric morbidity in a small fishing village in Northern Norway from January 9, 1939, to April 1, 1944, attempts to combine the genetic emphasis of the European investigators with the mental hygiene-environmental emphasis which he thinks is characteristic of some of the American studies. While making his observations his role was that of the local medical officer for the village during World War II. His findings, like Sjögren's, are quite detailed in breakdowns. Moreover, his figures are quite comparable as he reports an overall percentage of about 12.9 for psychoses and psychopathy combined and 5.56 for oligophrenia out of a total population of 1,080 persons over the age of 10 years. The figure would be reduced slightly if 245 children under 10 were included. He further goes on to examine the frequencies of the several types of psychiatric morbidity by occupation, race, migration, and wartime conditions.

His environmental emphasis is seen when he divides the total population into two groups, the secure group defined as self-supporting, 636 persons; and the insecure group described as on relief or spasmodically employed, 689 persons. He finds that the frequency of the psychoses are the same for both groups, the neuroses are more frequent in the "secure" group and psychopathy and oligophrenia are more frequent in the insecure group.

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In these two studies there is a definite attempt to present evidence showing genetic linkage although there is a marked caution in interpreting the evidence. The other striking feature is the close agreement of the overall frequency for psychiatric morbidity. In truth, if the fact that no frequency variations for the psychoses and oligophrenia in the different kinds of communities in the Western world could definitely be validated, then there would be every reason to suspect that any variation of these disturbances within the social structures of a given Western society are spurious and could not be explained by social psychological factors or processes.

Several hypotheses on a social psychological level have been developed. These hypotheses tend to accept rate differentials by geographical areas and/or social space and emphasize social factors that are supposed to be causative to a given mental disease or a group of diseases. Thus, if the validity of any one of these hypotheses could be established and it could be shown how the factor operates then one would have a most satisfactory interpretation of any rate differentials by time, geography or social space. Thus, the "social isolation" hypothesis, first proposed by Faris (11) and later developed in our joint work (12j) was most applicable to schizophrenia.

Since publication of MENTAL DISORDERS IN URBAN AREAS there have been numerous references to the "isolation hypothesis" in the literature but only three studies have appeared which provide certain data for appraisal. Lemert (27) attempts to give support to this hypothesis, as we did, by showing the correlation between first admission rates of mental disease and the percentage of various nationalities by counties in Michigan. His work added nothing and could hardly be taken as any test of the isolation hypothesis.

Jaco (20) in a somewhat more ingenious fashion attempts to develop a kind of index of social isolation by interviewing with a prepared schedule a carefully drawn sample of residents in four census tracts in Austin, Texas. Two of these census tracts had high and low schizophrenic first admission rates and

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the other two represented high and low manic-depressive first admission rates. He proposes nineteen null hypotheses to point up the differences in isolation between high and low schizophrenic census tracts. He rejects thirteen of these hypotheses and thus by this device infers that there is more social isolation in the high rate schizophrenic tract than in the low rate schizophrenic tract. In other words, he finds more schizophrenia in an area where persons show less contact and communication with one another. He, of course, does not show that persons who break down with schizophrenia are more isolated than those that do not.

This, of course, is just the point where Kohn and Clausen (22) begin their study, for they see clearly that if there is anything to the isolation hypothesis, it will have to be established through careful study of persons who develop schizophrenia as over against those who do not. They selected a sample of 45 schizophrenic and 13 manic-depressive first admissions to mental hospitals from Hagerstown, Maryland. They then secured a group of controls paired individually with the patients on the basis of age, sex, and occupation. The persons comprising the four samples were then interviewed through a schedule that covered the following topics: residential and occupational history, parental family relationships, friendships and activities of early adolescence, dating patterns, adult social participation, and a brief psychosomatic inventory.

They proceed to make careful qualitative analyses of their interview data and come up with the following findings: (1) About one-third of schizophrenic and manic-depressive patients, as compared to none of the controls, show evidence of being socially isolated at age 13–14; (2) there was no evidence that isolated patients were prevented from interacting with their peers because of lack of playmates, excessive morbidity, severe illness, or parental restrictions; and (3) no difference was found between patients and controls in their perceptions of family relationships.

The investigators recognize that their data are based on

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retrospective impressions of a group of patients but find also that their data secured from 26 out of 30 patients check favorably with the material in the hospital records. Their general conclusion is that "the data do not support the hypothesis that social isolation in adolescence is a predisposing factor in either schizophrenia or manic-depressive psychosis." They think rather that in those cases showing social isolation that this is an indication that a person's interpersonal difficulties are so great that he cannot very well continue to function in this area. Rather, it is a question of how he got that way in the first place so that he takes isolation as a way out. Further research along this line is very much needed utilizing other research designs for the study of isolation in the early years.

Gruenberg (16) in his preliminary report on an ecological study of the old age psychoses in Syracuse, New York, raises the question as to whether the patients who were not living alone were experiencing a process of social isolation and that effort should be directed to discovering if such isolation is symptomatic or causative.

Another hypothesis on the social psychological level has centered around the issue involved in the migration of peoples. The central questions are: Do persons who migrate from one place to another have a higher rate of mental disease than persons who live out their lives in a given community? If they do, is this higher rate caused by persons who are prone to a given mental disease moving around or is the higher rate caused by the fact that migratory persons are subjected to more severe stress than persons that have stayed home? These questions have been thought at times to be particularly relevant to schizophrenia. During the latter part of the nineteenth century much effort was expended by commissions and analysts to show that the higher rate of mental disease among foreign-born was due to the defective character of the biological stock represented by the immigrants. This position was very much undercut by Malzberg's work (29b) when he attempted to analyze first admission data by age, sex, nativity, race, and economic group.

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His results cast much doubt on the earlier position and at least serve to refute the notion that European countries were dumping their defective stocks on American shores. However, Malzberg's efforts cannot be taken as a disproof of the genetic factor in certain types of mental disease or for that matter a proof that rate differentials between native and foreign-born are the result of a difference in environmental conditions to which the different nationality groups have been subjected. Even so, it became somewhat fashionable in the 1930's to regard the higher rates of foreign-born and native-born of mixed parentage as due to the difficulties of adjustment to new cultural conditions.

Ødegaard (33) challenged this conception by his study of the Norwegians who had migrated to Minnesota. He interprets the higher rate of schizophrenic disorders among Norwegians who migrated to Minnesota as compared to Norwegians who stayed at home, as due to the fact that those who are more organically predisposed are most likely to migrate. But, like Malzberg, Ødegaard's evidence for the validity of his interpretation of rate differentials is inconclusive.

Tietze, Lemkau, and Cooper (44) in their study add no new dimension to the problem when they show that higher rates of psychopathy are found among those persons who move frequently as compared to those who reside for a long period in the same house. They found also that the rates were higher for intra-city migrants than for migrants from other communities. Again, one has difficulty in determining whether persons who migrate are more likely to be psychiatrically ill, or more likely to become psychiatrically ill because they migrate. One might develop a cultural integration hypothesis with regard to the above data. Persons who are firmly rooted and integrated in the culture of a community have a minimum probability of developing a functional mental disorder as compared to those who are not so well integrated.

Another hypothesis on this level is one that emphasizes the etiological role of a multiplicity of stress conditions in the social environment. This is a broad general hypothesis, less specific

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than the two discussed above, and stems in a large degree from the work of Faris and myself (12a). Here, our work was set in a broad theory of social organization-disorganization. We portrayed the city as having certain areas of marked disorganization characterized by cultural conflict, minimum consensus, slum dwellings, high population density, and high land values. The idea was that these areas of social disorganization produced disorganized persons and consequently more crime, delinquency, sickness, mental disease, and suicide was to be expected in these areas.

Leighton's Stirling County Study has been particularly designed to deal with the multiple environmental stress hypothesis. While the final report of this study is not yet available, there have been several accounts in the literature indicative of the trend that the research has taken. Leighton (25) himself has provided a statement of theory with some derived hypothesis that the research will supposedly test. Dohrenwend, a social analyst on the project, has provided a rather complete statement of the theory and objectives of the study. In line with the attempt to get at the etiological significance of certain socio-cultural factors, Dohrenwend states the central hypothesis, "that social disorganization impinges on such needs of the individual as those for physical security, sexual satisfaction, the expression and securing of love, the securing of recognition and the expression of creativity, thereby producing psychological stress and disruption" (7). Again in line with their general objectives, Dorothea C. Leighton (26) reports on the prevalence of psychiatric symptoms in a small town of 3,000 population on the basis of record searches and interviews. She reports that a much wider distribution of psychiatric symptoms exists than is commonly believed and estimates that approximately 37 per cent of the adult population in this town are psychiatric cases whether under treatment or not. This bears a close relationship to the study by Rennie and Srole (38) where they attempt to show the prevalence of certain psychomatic conditions in relation to social class.

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These last three hypotheses derive rather clearly from a type of social psychological theory. The hypotheses, broadly considered, can be viewed as inferences from differential rate distributions of mental disease, particularly schizophrenia. I turn now to a series of hypotheses which derive from what I have designated as the social system level. In this sense society is viewed as a functioning social system through space and time that sifts and sorts certain vulnerable personalities so that they get into (a) environments where the probability of a mental breakdown is increased or decreased, or (b) certain sub-cultural pockets that serve either to precipitate or induce a mental breakdown. The general hypotheses here can be designated as "social selection." In other words, certain persons because of age, sex, personality traits, intelligence, emotional instability, psychotic proneness, are selected for certain positions in occupational groups, city areas, marital status categories, institutions and the like in contrast to other positions in these structures as the social system moves through time. This process may be either active or passive as far as the person is concerned and through it one can account for significant differences in the rates for mental disease.

There are approximately four hypotheses that appear in the literature that represent various ways of stating the more general hypothesis of social selection. These four hypotheses are:

1. That certain persons because of personality inadequacies or mental disease proneness have a tendency to drift into certain social classes, sub-cultures or city areas.
2. That visibility of, and tolerance for, mental disorder vary with the attitudinal structure of different types of communities.
3. That certain persons because of their psychic needs to break their social ties tend to select and segregate themselves in areas, cultural or spatial, marked by anonymity.
4. That as the size of the city decreases rate differentials between socio-economic areas tend to disappear.

My intention at this point is to examine several of the more

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significant epidemiological studies in this area which have appeared during the last fifteen years and to show the manner in which these studies have called upon these hypotheses for negation or validity of the rate differentials that they have reported. In the earlier Chicago study by Faris and myself our procedure was primarily empirical even though set in the social organization-disorganization theoretical framework. We merely started out with the question as to whether or not the distribution of mental disorder would follow the pattern of rates that seemed to characterize the distribution of other social problems in the city (12b).

The contemporary research worker in this area, being more design conscious, generally sets up a series of statistical hypotheses which he purports to test for validity. This procedure is well-illustrated by Jaco's study of the distribution of mental disease in Texas (20) and the Hollingshead-Redlich study of the prevalence of treated mental disease in the class structure of New Haven (19a)[3].

Thus, Jaco begins his study with three hypotheses:

1. The probability of acquiring a psychosis is not random or equal among subgroups of the population.
2. Inhabitants of different areas exhibit different incidences of psychoses.
3. Persons with different social attributes or affiliations have different incidence of psychoses.

Jaco finds that his evidence gives support to his three central hypotheses. He is well aware that there may be a significant

[3] This study will, in all probability, become a classic for showing the influence of the class factor on mental disease rates. There are numerous aspects of this study that deserve comment, but our concern is with attempt to explain the rate variations by social class. The authors themselves recognize that social-cultural factors affect the prevalence of treated disorders in the population but do not represent essential or necessary evaluations in the etiology of mental disorders (p. 360). Dr. Redlich himself, to a question along this line, stated: "The New Haven study has not really brought out anything of etiological significance in explaining prevalence and prevalence itself is not a very good measure from an epidemiological viewpoint." (43). Further, it is of some interest that S. M. Miller and E. G. Mishler in their expository review of this volume, decide not to discuss the issues raised concerning the relationship of social factors to the etiology of mental disease, but rather to consider the study's implications for psychiatric practice. (31).

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gap between "true" and "treated case" incidence. However, his findings might as easily be interpreted through the general social selection hypothesis as they can be with respect to "industrialization," "anomie," and "enculturation." The interesting fact that his findings in certain instances run counter to the findings of other studies should give one pause. For example, his high rates for both males and females among professionals and semi-professionals cannot be easily explained away by the "marginal status" supposedly enjoyed by this group in Texas. Again, his low rates among Spanish-Americans for both males and females are unexpected and contrast rather sharply with the high rates frequently reported for various ethnic groups in northern states. This might be caused by the fact, as Jaco suggests, that the Spanish-Americans are not well-integrated into the dominant Anglo-American group and also have a very protective kinship system. It may also be explained by the hypothesis that the visibility and tolerance for mental disorders in this group is at a variance with the dominant Anglo-American group. Again, much more research will be needed to show that a closed, intact, integrated group has less mental disorder than one that is less integrated around a common core of values. Eaton's findings for the Hutterites might prove to be an example of a negative case.

Now, the point I wish to make is that when Jaco's findings run counter to findings of other studies, like other investigators he tries to account for them by social factors even though the total evidence is contradictory. His hypotheses are broad statistical statements, which, while no doubt true enough in terms of his evidence, provide us with no basis for accounting for the rate differentials as found in age, sex, marital, occupational, or ethnic groups. He could just as easily have used some hypothesis centering on "social selection" which he seems fairly close to doing when trying to interpret the lower rates among the different age and sex categories of Spanish-Americans.

Let us examine now the Hollingshead-Redlich hypotheses, also three in number:

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1. The prevalence of treated mental illness is related significantly to an individual's position in the class structure.
2. The types of diagnosed psychiatric disorders are connected significantly to the class structure.
3. The kind of psychiatric treatment administered by psychiatrists is associated with the patient's position in the class structure.

Much the same sort of criticism can be centered on this study of social class and mental illness. While their organized data give support to their hypotheses, their study is largely of prevalence and our concern must perforce be with incidence rates, for these should give some clues as to whether social factors bear some relationship to the etiology of mental disease. Here the expectancy would be that if social class factors have some relevance for etiology one would expect that incidence rates for a given disorder would have a significantly inverse relation to the class structure. Further, one would have to establish that the higher rate of persons in the lowest class is made up of persons who originated in that class. This is under the assumption that the conditions of life at a lower class level in our society are more likely to be psychotic-inducing than other kinds of life conditions enjoyed by other social classes.

Now, Hollingshead and Redlich do have several tables of incidence rates constructed from those persons who entered treatment for the first time during their six months observation period. When they compute the rates based on these cases for the four social classes, they find, using the chi-square test, that the difference is significant at the 5 per cent level. However, the rates do not vary inversely with class as the following table[4] shows:

Class	*Rate*
I–II	97
III	114
IV	89
V	139

[4] Source: Text Table p. 212, Social Class and Mental Illness.

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These data are, of course, based on all the cases of mental illness that entered treatment for the first time. When this total group is broken up into neurotic and psychotic groups, the incidence rates for the neurotics do not vary with class structure. Here, Class IV has the lowest rate and Class III the highest rate with Classes I–II and V having rates of 69 and 66, respectively. However, in the case of the psychotic group the incidence rates vary with the class structure but the differential in the first three classes is probably not significant while Class V rate is twice that of the other rates. This might indicate that Class V contains many more psychotics than might be expected provided that they started in Class V. This the authors later try to show in discussing the drift hypothesis. Their Table 17 shows that schizophrenic incidence rates vary with the class structure although there may be questions of whether or not the differences between the classes are significant or the case basis for the classes is sufficient for claiming a difference.

In this study these data, as presented above, might constitute the evidence for arguing that the influence of life conditions on a given class level plays some role in the development of a particular psychosis. But the case is not proven beyond any doubt. The concentration of the higher incidence of psychoses in Class V might just as easily be regarded as due to some form of social selection—a hypothesis which is not stated in their work. The denial that the neuroses are linked to class (19b) does not quite stand up as the prevalence rates for neuroses vary directly with the class structure and is certainly a function of the fact that the upper social levels in New Haven society have more money to spend on this type of personality perturbation.

In one of their articles, Hollingshead and Redlich suggest a social selection hypothesis when they state that "current prevalence is a measure of the responses patients in the several classes make to the treatment process." This, they label as the "differential treatment hypothesis" (18).

Some data from England serve as a challenge to the New Haven study. In an address before the British Sociological So-

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ciety, Dr. J. N. Morris (32) presented the following rates for England and Wales.

First admissions for schizophrenia (per 100,000 men age 20 and over) in 1949–53 in the five social classes.

Class	I	II	III	IV	V
Rate	51	57	93	103	229

He then asks the ever-present question "Is this fact caused by a downward drift of men to Class v or by the social and familial environment in Class v that tends to produce an excess of schizophrenia?" He then points to a study that has classified a national sample of schizophrenics, ages 25–34, by occupation and social class and then classified their fathers by occupation and social class on the basis of an examination of birth certificates. The answer was clear. There was an excess of schizophrenics in Class v but their fathers were distributed rather evenly over the five classes. This seems to indicate that the patients experienced a downward drop because of their illness and to explain their excess in Class v. Thus, here in this study, the schizophrenic illness operates as a selective factor or, from the opposite angle, the social system functions to place these men in Class v.

Lilli Stein's study of class and schizophrenia (42) is relevant here because her findings also contradict some American results. She proceeds by selecting four East London boroughs (E) outstanding for their high proportion of men in Classes IV and V and five West London boroughs (W) outstanding because of their high proportion of men in Classes I and II. She then tabulates the first admissions to mental hospitals from E and W boroughs for 1954–1955 by age, sex, and three diagnostic groups—schizophrenic, manic-depressive and psychoneurotic. The results run contrary to those found for American cities. Her findings showed that the W boroughs had significantly higher rates in practically all age and sex categories as compared to the E boroughs. This held true, in general, for schizo-

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phrenia and psychoneuroses; it was less marked in the manic-depressive group. She then proceeds to distribute these data among the social classes in the two sets of boroughs. While the rates are still higher in W she does find in both areas that they vary inversely and consistently with the class structure and for all three diagnostic groups. While she concluded that there was a real class gradient in the inception of schizophrenia she thinks these results must be qualified because of the radical differences between the E and W boroughs in the sex ratio, number of persons living alone, types of private households and origins of the population. These differences signify that a single index of social class may not be too meaningful.

In another study from England, Carstairs and Brown (3) attempt to get at the incidence and prevalence of psychiatric disorders in two different types of communities, Rhondda, a coal mining region, and Vale, an agricultural area. Rhondda had a more densely concentrated population than Vale which was rural and spread over a wide area. Rhondda was found to have more psychiatric cases than Vale (3.8 to 2.6 per 1,000 population). They also found that in Rhondda psychiatric cases were more heavily concentrated in the non-miner group. This might be regarded as an instance of the vulnerability of a minority group but the investigators are more inclined to think it is an example of adverse selection. They conclude that the linking of social pressures to psychiatric disorders must wait upon an analysis of the social structure and value system of a community, the circumstances leading to the emergence of declared cases and the carrying of undeclared cases in each community and the clinical features of the illnesses.

Ødegaard has been one of the most constant proponents of the social selection hypothesis. From his earlier study of emigration (33) where he emphasized the tendency of the psychiatrically vulnerable persons to migrate to his more recent analysis of psychiatric cases in relation to the occupational structure of Norway, (36) he has generally attempted to show that social selection versus environmental stress provides the

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most enlightening explanation for the rate differentials in various social structures. His study of the incidence of psychoses in the various occupations analyzes 34,457 first admissions to Norwegian mental hospitals, 1926–1950. In general, he finds the highest admission rates among those occupations with the lowest social prestige. He finds the highest rate in the seamen of the merchant marine. This holds for all psychotic groups with the exception of epilepsy, mental deficiency, and manic-depressive psychosis, although in these diagnostic categories the seamen are second from the top rate. He explains this by a social selection process as well as a secondary selection which takes place when men around 30 seek other occupations leaving the unstable and psychopathic types in the seaman's group. His finding that the high rates of manic-depressives are in the more favored occupational groups is in conformity with American results, but the mechanism of social selection is more evident for schizophrenia and for the psychoses with epilepsy and mental deficiency. He also thinks that social stress and protection are not explanatory of the distribution of psychoses in the occupational structure.

It is of some significance to note that the hypothesis of social selection is frequently urged when dealing with such special structured institutions as the army, merchant marine, or prison. Winston in her study of psychoses in the army (45) asks "Does mental disease act as a further selective factor in the already highly selected military group?" While she reports no tendency for mental disease to increase in the army, she does find that the psychological misfits are weeded out eventually so that mental disease tends to decrease as length of service increases. This was the opposite of Ødegaard's findings with respect to the merchant seamen but probably means only that the seamen make a voluntary choice to leave the service while the army actively weeds out those who cannot fit into the rigors of army life.

Sims (40), in a study of noncommissioned officers in the British Army, Dominion Army, and prisoners-of-war, also sug-

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gests that the difference in the incidence of the disease in the various groups is due to high selection standards. He further reports that there is, in addition, a secondary selection as many recruits are weeded out in basic training and this lowers the incidence among those who get overseas. Ekblad (10) also supports Ødegaard's social selection hypothesis when he shows that his sample of seamen have higher rates for psychopathy and schizophrenia when compared with non-seamen in naval training.

Now, I have pointed to these studies to illustrate the application of the social selection hypotheses when applied to certain rate differentials in selected social structures. It seems to be much clearer in application when dealing with occupational, marital status, and specific institutional structures than it is when applied to social classes or geographical areas. In the latter, the problem is much more complex although even here the social selection hypothesis must be considered. In essence it is an hypothesis that explains significant rate variations as due to the manner in which a given social system functions through time and in its functioning tends to sort and sift persons into class and community positions.

As we indicated above there are a number of more specific hypotheses that stem from this general hypothesis and these I now wish to examine. Both the "drifting" and "segregation" hypotheses are variations of social selection. "Drifting" implies an involuntary segregation and results from the automatic functioning of a social system through time. "Segregation" implies a voluntary, conscious selection of a preferred place of residence in a community. The "drifting" hypothesis came into focus over twenty years ago as an attack on the rate distributions for the various psychoses in Chicago and Providence that Faris and I (12a) presented in our joint work. It was quite a theme for some reviewers at the time for it questioned the significance of our rate patterns. At the time we argued that the concentration of the younger catatonics and paranoids were quite similar to the concentration of older cases and this fact argued against

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the thesis that the pattern could be explained by "drift" alone. This issue was examined by investigators by locating the addresses of a sample of schizophrenics from Buffalo twenty years prior to their first admission to state mental hospitals during 1949–1951 (24). They concluded that the concentration of schizophrenic cases in low-income areas was not the result of downward drift from better areas. They further showed that the high rates are not caused by the drift of men living alone.

Hollingshead and Redlich (19a) in their study also faced this issue since much of their results would be dependent upon the fact that mental patients are not excessively mobile by class. They examined for their schizophrenic group four sets of data —the nativity of schizophrenics compared with the nativity of adult population in the community; the birthplace of native-born schizophrenics in relation to class; the past addresses of schizophrenics, and the class position of their families of orientation as compared with their own social class position. From these data they concluded that there was no evidence of a downward class drift of schizophrenics. In fact, the evidence showed that 91 per cent of the patients were in the same class as their families of orientation. These data raise certain questions about the class mobility of patients. What is the class mobility of the residents of the community itself? Are schizophrenic patients less mobile by social class than non-schizophrenics in the community? One might well expect that they would be but how then does one account for those observations that schizophrenics are more likely to be geographically mobile than non-schizophrenics.

Prior to the Buffalo study, Gerard and Houston (14) on the basis of studying an ecological distribution of male schizophrenics in Worcester, Massachusetts, concluded that the high rates of schizophrenia in the low social-economic areas are to be largely explained by patients living alone. They found that patients coming from families showed marked residential stability and those patients not in families showed marked residential instability. They suggest the hypothesis that these non-family

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men used residential instability as a means of protecting themselves against involvement in disruptive family relationships. Thus, they imply the segregation hypothesis.

Hare (17) uses this hypothesis to explain the concentration of schizophrenic cases in Bristol, England. He starts out by trying to check on our findings for Chicago to determine if there is additional evidence for supporting the social isolation hypothesis. He shows that, like our findings, the schizophrenics were concentrated in the central areas while the manic-depressives were more widely distributed. He points out that the high rate in both "good" and "poor" central areas is related to the factor of living alone—thus supporting Gerard and Houston. He also raises the alternative, namely, that the concentration of schizophrenics is to be explained either by segregation or the causal effect of the environment.

Another hypothesis that is a variation on the social selection theme stresses the differential visibility and tolerance of areal populations for mental abnormality which, in turn, accounts for rate differentials in different communities. Mary Bess Owen (37) first raised this question years ago when suggesting that this hypothesis might explain our contrasting distributions of paranoid and catatonic schizophrenics. In a more recent study (2) there was an attempt to test the hypothesis that if a difference in demand for hospitalization causes higher urban rates, the urban excess should be predominantly in the rate for cases whose symptoms are sufficiently tolerable socially that care outside of a hospital would be possible. They then proceeded to examine the rural and urban first admissions in Western Ontario, broken down so as to give rates for severity of symptoms for age groups 45–64 and 65 and over. Their results showed that for the age groups studied the excess urban admission rate is almost entirely explained by the greater urban tendency to hospitalize cases whose symptoms would be tolerated in a rural community. They conclude that this device may be useful in distinguishing between the real and apparent difference in rates of mental disease.

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Finally, there is the hypothesis that as the size of the city decreases the rates tend to approach a parity for the different socio-economic areas. This proposition was demonstrated rather sharply by a study (5) of the distribution of first admissions of schizophrenics in Hagerstown, Maryland. The findings are extremely pertinent for they contradict the Chicago findings and by implication raise questions about the spurious quality of the rate differences found in a large city. The findings show that there is no difference between the schizophrenic rates in Hagerstown and the remainder of the county and no difference between the rates in five areas of Hagerstown arranged on the basis of a rental value index. They also show that the absence of areal rate differences cannot be explained by shifts in diagnosis or unusual upward or downward mobility. Five possible interpretations of these findings are offered but the one favored by the investigators is that the differences in socio-economic areas of a small city, like Hagerstown, are not sharp enough to show the differences that the large cities produce. They conclude with the question: "In the constellation of values, attitudes, behaviors, and relationships that are generally indexed by socio-economic status, by occupation, education and area of residence, what are the factors that are crucially related to schizophrenia?"

In this review of social structures and mental disorders, I have attempted to concentrate on the various hypotheses that purport to explain significant rate differentials in selected social structures. The fact that the rate distribution in different social structures varies for the different diagnostic groups makes the attempt to interpret total mental disease rate differentials as of doubtful etiological value. From an environmental perspective, interest is greater for the so-called functional disorders but even here the evidence is highly inconclusive for asserting with any confidence that a high rate in a given position of a social structure is a product of certain stresses, strains, and conflicts in that position.

I have also attempted to show that certain basic methodologi-

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cal orientations about the nature of man in human society enter into the way one views these rate differentials. This is partially reflected in the contrast between the European epidemiological studies and the American ecological studies of mental disease, and between those investigators with a biological orientation and those with a sociological orientation.

Biological, social psychological, and social system theory have been productive of various hypotheses to account for significant rate differentials in social structure. Biological hypotheses have largely been interested in the comparative count of psychiatric cases in different population groups. Here, rate differentials are to be explained away. In contrast, social psychological hypotheses are likely to assume that rate differentials have been reliably established and then to validate the hypotheses by other research designs. To date, the results on this level have been inconclusive.

On the social system level, the hypotheses have been largely directed to showing how the rate differentials are functions of the manner in which the social process works in communities and social structures. Here, one has certain sociological hypotheses that give us added knowledge about the social system but are not stated in a form that can show the relevance of socio-cultural factors for the production of specific mental abnormalities. The task which the social scientists face here is to be able to show that certain stresses from a given position in a social structure make such an ingression into a given human experience that his mental content and behavior emerge in such bizarre forms that he cannot be fitted into the social structure at any point. Then, too, from the standpoint of prevention there is a need to know the positions in the social structure to which the more bizarre behavior types can become adapted. Probably, it is not without point to note that man in addition to trying to correct certain behaviors and make them more acceptable, must also learn to live with the type of personality structure that, through events and experiences, he has made for himself. In any event, the task of the social scientist in this

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area is not likely to be facilitated until the clinician has developed methods for isolating those persons who will not break down with a mental disease no matter how extreme the adverse, traumatic, or discouraging elements in his life will be. On the other side of the ledger, however, it may be that these epidemiological studies of specific mental diseases may provide some clues for distinguishing between different kinds of abnormal personalities that are caught up in the network of a present diagnostic category.

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DISCUSSION

DR. DUNHAM: I thought I might indicate some of the assumptions that I had in mind in writing this paper. I hope that it has agitated the rest of you as it has Professor Jaco. We were having a discussion about it. It is a great subject of controversy.

What I had in mind, of course, is the strikingly contradictory interpretations of various ecological and epidemiological studies dealing with mental disease. While there are contradictory findings, there are many more contradictory interpretations of these data.

I want to say a word about my assumptions. I am assuming that schizophrenia eventually will be more reliably diagnosed. What con-

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stitutes schizophrenia now obviously covers a multiplicity of reaction types. We want to separate out these types—I mean some sort of a core group as over against other types now called schizophrenia.

The second point I would like to make repeats one that was made earlier this morning about the social values in the community—those significant values surrounding the patient or person who goes to a treatment facility and which determines entrance into the facility. While this may be true, it says nothing about the different degrees of pathology, or disorganization, or disturbance, which these patients may have.

It seems to me this is fundamental, because when we talk about these values we are really talking about the differential tolerance of different community settings. While this is all right to talk about, it is something separate from the pathology of the individual patient.

The third point is that I am taking for granted that epidemiological studies should go on. If we develop some more ingenious designs, we may be able to throw some light on differences in schizophrenic groups and, also, we may be able to get some leads in etiology which might be investigated by other methods.

DR. ERNEST M. GRUENBERG: Because of a last minute cancellation by the invited discussant Dr. Morton L. Levin, it has fallen to me to prepare some introductory remarks to start the discussion. I found great difficulty in disagreeing with Dr. Dunham's paper but by applying myself thoroughly to the task I have managed to find a few points which I think need to be made.

The first concerns the two definitions of ecology and epidemiology, where ecology is defined as the study of the environment and life history of the organism in contrast to epidemiology, which is called the sum of what is known about epidemics. I don't think this is a very useful distinction, for hardly any existing epidemiologist would define epidemiology so narrowly.

Although not all would accept as broad a definition as John Gordon gives, "epidemiology is medical ecology," I think many of us would accept the notion that this is a fair approximation of the correct definition. This, then, would make epidemiology a special field of your ecology. But since the purpose of getting together is to review our state of knowledge regarding the causal factors which affects the occurrence of mental disorders, it is not really necessary, I would say,

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that we agree with one another as to which is the most appropriate word usage; by your definition we are all ecologists here.

It is clear also, I believe, that those who are preoccupied with the physical features of the environment or with the gene characteristics of the organism, are well aware of the importance of the social and cultural environment and, contrariwise, those preoccupied with the social environment appreciate the importance and relevance of the physical environment.

As far as I know, no one in this room is an advocate of a brainless psychiatry, or of a mindless psychiatry, or of an asocial psychiatry.

Toward the end of your classification of the uses and difficulties of doing these studies, Dr. Dunham, you raise the possibility of getting out of some of the difficulties by considering, as cases of mental disorders, persons who are assigned this characteristic by the social environment, using what some people call the social definition of mental disorders.

I really don't think you mean to say that this concept of a social definition or identification of case is equivalent to the cases who, in fact, come to clinical attention as patients of psychiatrists, even though I got an implication from the text that this meant that there was no other mode of socially identifying a person as mentally disordered.

Further on, you discuss the use of biochemical identifiers, or an analogue of biochemical identifiers, as a way of getting around the problem of case selection for research.

Here again, I felt that I couldn't go along with you all the way as it was written, because I don't see how such an identifier gets us out of the problem that we are confronted with. For any such identifier must be validated. First, the identifier has to be found, and to do that you have to discover what proportion of your cases give positive results, and what proportion of those giving positive results are real and not false positives. To do this you have to have an independent set of criteria regarding the nature of the case.

I don't see how such identifiers would save our souls in any way. For it seems to me that there is no general answer to the question, and any particular study is quite justified in selecting the clinical entity it wishes to investigate. The suitability of the criteria used for identifying cases depends, first, on the relevance to the entity to be studied and, second, on whether or not the criteria used introduces

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bias in locating and identifying cases in the sub-samples of the population which they intend to compare.

For example, taking the very simple classification by social classes, will the criteria give a higher proportion of the cases existing in the population in one social class than in another social class? Is the method of case-finding and the criteria applied biased with respect to the variables that they are going to be analyzed by?

In the discussion of Ødegaard's and Malzberg's data regarding the availability of beds in hospitals as related to the variation in mental hospital admission rates, I got the impression that you used their data and their arguments against their idea that mental hospital admission rates might be a function of bed availability. Both Ødegaard and Malzberg give examples where the increases in rates that they are pointing to do not coincide with a greater number of beds per capita in the population at risk. However, it seems to me that the concept of availability of beds is much broader than the number of beds per capita existing within a jurisdiction; surely there are other meanings of availability, some of which you mentioned earlier. I couldn't see why you were so easily persuaded by this measure, for it seemed to go against some of your earlier arguments.

In reference to the Hagerstown studies by Clausen and his associates on social isolation, I wondered if you would comment more on two features of those studies. One of the things that is peculiar about their findings is that there wasn't any difference in the manic-depressive cases and in the schizophrenic cases with regard to the social isolation found in youth. I would be interested to hear your discussion on the theoretical implications of the failure to find differences.

The second part of this study[1] (which I agree is of great importance to the field you are discussing), although it found no differences in social environment factors of the youths who later became cases, they did find that the cases had had less social interaction. Would not this in itself, at the age of 13 or 14, regardless of the causes, have later effects which would fit into some of the theoretical concepts that you have advocated in the past? Could not this fact be regarded as a causal factor even if the cause of their failure to take opportunity for social interaction was not clearly understood?

[1] Kohn, Melvin L.; Clausen, John A.: Social Isolation and Schizophrenia. *American Sociological Review,* 1955, 20: 265–273.

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The last point I will bring up at this stage is that while you raised a number of models for explaining the concentration of cases of schizophrenia in various sections of the population—usually in the center of cities—and while you described various features of the different drift processes, there was one model that has been suggested which I didn't find, but perhaps in my haste I might have missed it. This is the notion that most people who spend the early part of their lives in the center of our cities move out of them by adult or early adult life, and that the central concentration of cases is due to their failure to migrate away from the center rather than because of their migration to the city's center. Such a model proposes that in a socially mobile society the standard pattern is mobility upward and, therefore, the concentration of cases occurs as a residue rather than as drifting into lower social groups. It seems to me that the analysis of the data within the framework of such a model would give us slightly different results.

The last point that I wish to discuss (and which isn't original with me), is that upward mobility may, in fact, be a protector against the development of some of these syndromes; that this relationship accounts for the high concentrations of cases which are found in an environment unfavorable for personality development which increases the risk of developing schizophrenic psychosis. This kind of hypothesis, it seems to me, would be very easy to test.

The easy way to test it would be to take a random sample of people living under undesirable conditions and split them in half, giving one-half of them a lot of help in moving upward socially and comparing their experience over the next ten years with those to whom one did not give a lot of help.

Finally, I thought I would like to mention one administrative implication which seems to me quite clear. As with so many other health problems, much of the mental disorder data which Dr. Dunham reviewed shows a high concentration of pathology in that part of the population which has the least financial resources to pay for help and for preventive activities.

This relationship once again, as it has done so many times previously, raises the question as to why our physical and mental health services should be operated on the basis of the ability of lower income groups to finance these services, when, in fact, we know that those who can produce the most in the way of taxation have the least need

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for the services, while those who have the least ability to raise the tax money have the most need for services. In other words, the whole doctrine of the development of health services out of local resources seems to me to be incompatible with the kind of data that has been presented here.

In closing, I would like to thank Dr. Dunham for a very interesting and stimulating paper.

SUMMARY OF DISCUSSION

1. Movement in an urban environment, particularly at the present time was classified into two types: the first being intentional movement, the second being forced.

Individuals or families might elect to change residence because they had moved up in the social scale or for some other reason. Forced movement, on the other hand, could occur under such circumstances as urban renewal, where families had to leave neighborhoods where their roots might have extended back for years, even generations. A study of both these types of movement might throw light on some of the points raised earlier on stresses, on how different types of people in each of the two groups handled their stresses, and what the consequences were.

2. Issue was taken with Dr. Dunham's view that epidemiological studies of mental disorders support theories of genetic origins when the statistical differences are at a minimum.

It is well established that, in many diseases where a genetic factor has been demonstrated, considerable rate differences between populations occur. These differences result from the several factors that influence gene frequencies in populations, such as migration, genetic drift, and selective fertility.

Genetic explanations, where rate differences between population are small, as in schizophrenia, suggest that whatever genes are involved are rather widely distributed and, therefore, not as subject to drift, migration, etc. However, one instance investigated by Dr. Böök showed a 3 per cent rate in a population in the north of Sweden as against a 1 per cent rate in a population in the south. This appreciable difference, based on carefully checked diagnosis and apparently representing a true variation, cannot be taken as evidence against genetic etiology. They can be regarded as suggesting that the

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living conditions and culture in the north favor survival of people with the relevant genetic makeup more than do conditions in the south.

3. Further discussion on the implications of findings regarding differences in the incidence of a given disorder, such as schizophrenia, in different populations and different cultures, emphasized the importance of the investigator's basic approach to these data. One approach seeks out constancy in incidence rates and thereby justifies fatalism regarding preventive work, while the other, which looks for variations in these rates feels it can justify a belief that preventive manipulations might bring high rates down to the lower levels of the low rates. Concomitantly with this line of reasoning, is the tendency to suggest that constancy of rates implies a biological origin of an illness while variation in rates a social origin. Strong issue was taken with one or both of these inferences. However, no disagreement was expressed over the statement that, since these assumptions did affect so many people, findings emphasizing variation or constancy did tend to affect practice.

4. Dr. Dunham in his paper suggested that many were skeptical of rate differentials of mental illnesses which depended on first admission rates to mental hospitals. Dr. Kramer, however, emphasized that variations in first admission rates were well established but that disagreement arose over their interpretation. "True" incidence must account for both first admissions hospitalized during the index year plus the new cases which developed during that year but which were not hospitalized. It was this last factor—the new cases which were not hospitalized—which created difficulties.

Dr. Kramer suggested that the problem be examined in the following manner. If:

Iy = "True" incidence rate for index year (y).
Cy = All cases developed during year (y).
Ry = First admission rate for year (y).
Ay = Cases developed and first admitted during year (y).
Ax = Cases developed prior to year (y) but first admitted during year.
Az = Cases developed during year (y) but not admitted during year.
P = Population at risk during year (y).

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$$Iy = \frac{CY}{P} = \frac{Ay + Az}{P}$$

Then:

$$(I). \quad Ry = \frac{Ay + Ax}{P}$$

In formula (I), first admissions are separated into those cases who developed during the index year (Ay), and those who developed any time before the index year (Ax). Now, if the latter group (Ax) were to be replaced by the group of cases which developed during the index year but were not hospitalized during that year (Az), the numerator would become the total of cases who developed during the index year (Cy); in other words, this is the numerator needed for finding the "true" incidence rate. In effect, the first admission rate departs from the "true" incidence rate by substituting cases who were admitted during the index year but who developed prior to that year (Ax), for those cases who developed during that year but who were not admitted during this period (Az).

Dr. Kramer then performed the following manipulation on formula (I):

$$Ry = \left(\frac{Cy}{Cy}\right) \cdot \left(\frac{Ay + Ax}{P}\right)$$

(multiplying the rate by a fraction equal to unity does not alter the rate)

Producing the following formula:

$$(II). \quad Ry = \left(\frac{Cy}{P}\right) \cdot \left(\frac{Ay + Ax}{Cy}\right)$$

Since the "true" incidence rate for the index year (Iy) equals the total number of cases developed during the year (Cy) divided by the population at risk (P), we may substitute, Iy for $\frac{Cy}{P}$

$$(III). \quad Ry = (Iy) \cdot \left(\frac{Ay + Ax}{Cy}\right)$$

Thus it is evident that the first admission rate (Ry) is a function of the "true" incidence rate (Iy) multiplied by a factor which relates the total number of newly developed cases (Cy) to the number of cases who were new admissions (Ay) and the number of old cases

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who were first admissions (Ax). This factor can be called the coefficient of the "true" incidence rate in the first admission rate; or, more simply, "the coefficient."

But in comparing two different situations the difference between the two coefficients might be large enough to severely distort, or even reverse, the rates being compared. Was there, Dr. Kramer asked, really a higher incidence of schizophrenia amongst single men than among married persons as admission rates seemed to indicate? Or was this merely due to the protective influence of marriage which was more likely to keep a case at home rather than to throw him into the hospital?

While in all the published works of Malzberg or Ødegaard various explanations were given for observed difference in admission rates, no data were offered to support the validity of the difference with respect to true incidence.

In all epidemiologic studies that dealt with hospitalized populations, it was considered important that true incidence and first admissions be related. Otherwise the interpretation of the observed rate differentials had to remain dubious.

5. This view, in turn, was attacked in the belief that a true incidence rate could never be attained. Dr. Dunham's paper considered that the social definitions of who ought to be hospitalized affected the rate at which the people were hospitalized, implying that it was necessary to penetrate the effects of these varying definitions in order to get at the picture of true incidence. But what was considered to be a suitable case for hospitalization, and how people considered disease, affected the course of the disease. From this point of view the problem was not one of weighing the relative effects of pathology on the one hand, and of social tolerance on the other, but rather of appraising the effects of social attitudes on the development of pathological reaction patterns.

If this were the case, then the "true" incidence rate, in the terms of the previous discussion, was, in fact, unattainable.

DR. DUNHAM: I want to thank the discussants for their critical remarks, and I think I will start at the middle and work around to Dr. Gruenberg, because I think Point 3 hits at what is a crucial aspect of the formulation.

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It was said that if you take the position that there is a uniformity of incidence among the various peoples throughout the world, then this fact would discourage the search for methods of prevention. I am not so sure whether I would agree with that, although I do think that if you could establish that there are marked differences in incidence of mental illnesses among peoples throughout the world, this would encourage a search for sociological explanations of mental disease, as over against biological formulations.

On the other hand, I agree that these things are not completely mutually exclusive because the human biological organism always interacts in an environment and culture.

Even if one accepts this, it seems to me that there would still be various therapeutic possibilities, even though a given mental disease might be distributed fairly evenly among the populations of the world.

The very last point, Point 5, is very intriguing to me because in some of my own papers I have argued in the same way: that how people conceive of a person's behavior or syndrome, and so forth, will affect the process or the course of the disturbance. In fact, one might argue that this is in some ways an operational definition and that if we could really measure these judgments then we would not have to look further: this *is* the disease. If I have shifted my position, it is probably because of various kinds of influences and new evidences that have been brought to my attention.

On the other hand, as I said in my opening remarks about my assumptions, that while I agree that this may be true, at the same time there may be a biochemical basis for certain of these diseases, but you could still have the course of the disease being affected by the attitudes and values that are prevalent in the community.

We probably will come back to that again. I don't think that the issue is settled by any manner of means, since it represents two ways of looking at the same phenomenon.

I think that I agree with everything that Dr. Kramer remarked about the interpretation of hospital admission rates (Point 4), though I will have to examine his formula more carefully. I think that his comment on skepticism over the significance of rate differentials is borne out by the whole paper, for I am skeptical of the interpretations too, regardless of whether we can get true incidence or not. The whole paper is concerned with this matter of evaluation and interpretation, particularly contrasting those of the social scien-

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tists with those of the medically-trained people. It strikes me in reading the literature that the medical person in his type of study has always been able to say that other factors account for the difference as found; while if you move to the Hollingshead and Redlich type of study, they are trying to show that social class factors in some way affect the incidence and prevalence of the behavior.

Moving on to Point 2—the discussion about the relation of genetic theory to unvarying incidence rates—the interpretations are the same as mine, if I understood them correctly. I was saying that if there are no statistical differences or if they are at a minimum, this would imply an acceptance of the biological position and in this I did not take account, (and it is quite true) of the possibility of gene drift or of the selective factors in reference to genes in the population. I have sometimes argued—and Dr. Böök is the authority here, and I am not—that in genetics you ought to have a random distribution of the disease throughout the population whereby if there was something to the role of social factors, or stress factors, or cultural conditions as affecting the incidence of mental disease, then the disease would not be randomly distributed throughout the population.

I agree with the statement about spontaneous and forced migration (Point 1). It certainly would be interesting to make a comparison between those who move because they want to improve their situation, as over against those who are forced to move. Also it seems to me there is a third category—those that seemingly just drift or never get started any place. This was somewhat the burden of Dr. Morris' interpretation in England, and his findings, which contradict those of Hollingshead and Redlich, have already been referred to. One interpretation was that the fathers, who are evenly distributed by social class, had sons who never got off the ground, so to speak. These sons were counted in the lower class position because they never seemed to have the push, or the energy, or whatever it may be, to do something to change their situation.

To come to Dr. Gruenberg's opening remarks. First, we might speak of biochemical identification. I am not a biochemist, of course, but I am informed from various sources that there are supposed to be advances on this front. However, when we examine the evidence it often seems to go up in smoke and we have had many disappointments. At the Lafayette Clinic they seem to think they are close to a biochemical breakthrough. I don't know.

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Dr. Gottlieb, head of the Clinic, sometimes tells me "If we can perfect some biochemical identifier, why, we will have a more objective designation of these cases, and we will have a sounder basis for an epidemiological study."

I don't know whether any biochemical test will ever be perfected or not, but it has struck me—and I speak primarily of schizophrenia —that if this should happen, it would certainly seem to me—and I know it has been the dream of the people in this area—that you would have a device for saying "These cases respond positively to the test, but those do not." Those schizophrenics who do not respond to the test—those we have been calling schizophrenic—are probably adjustment problems. We would then have an opportunity of making comparisons between this group and the ones responding to the test, assuming other etiological factors or forces would be operating in the negative group. At least, that was the way I tended to look at the thing.

You asked me about the Hagerstown studies, referring particularly to the one by Clausen and Kohn on isolation in schizophrenia, where they found that one-third of the manic-depressives and one-third of the schizophrenics reported the same amount of isolation in their early adolescent years.

I think that was interesting, despite its defects as a retrospective study. It is a backward glance, of course, but Clausen and Kohn seem to try to show (and they spent a lot of time doing this) that in the situation of these patients there was no reason for them to be isolated in the sense that they did not have any playmates or as many people surrounding them as the persons that were used as controls. The situations of both study and control groups were very similar.

Therefore, when you raise the question as to why they were isolated, you will again have somebody here who will say that, whatever is the matter with the person, and whether or not he does go out and interacts, he will probably tend to withdraw and be by himself, consequently other people will leave him alone. To account for why the manic-depressives and schizophrenics are the same, I have no interpretation here. Probably there was something the matter with their diagnosis! I remember when I was doing a study years ago on catatonics and brought some of the recovered patients to be examined, the psychiatrist invariably changed the diagnosis and said they were manic-depressives.

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Dr. Gruenberg also made a remark toward the end about the possibility of upward mobility: that this is one of the values prized in our particular type of society, and that it might be preventive of disturbance.

I was reminded of a theoretical paper that Warner gave 20 years ago when he argued the reverse: that striving upward may produce a kind of tension. This idea was quite popular during the 30's and as one looks back on a lot of that literature, one wonders how much it was frequently bound to the particular social, cultural, and psychological climate and situation of the period. Those interpretations really could not be supported by any hard and fast evidence; so that while one might make a proposition one would be able to test, it strikes me—to refer back to Morris' study—that many of these schizophrenics may not get started upward at all because of the nature of their disturbance.

Perhaps this will show that I have tried to respond at least to all of your comments, and that I appreciate your remarks.

CHAIRMAN LEIGHTON: Thank you, Dr. Dunham.

I will not try to summarize the meeting today. There have however been a number of general themes that seem to run through the various papers and the discussions that might be useful to point out; and I may also take this occasion to slip in some reactions that have been turning over in my mind.

One matter is the problem of recognizing the phenomenon with which we are concerned, psychiatric disorder. This involves the conceptual and methodological question of standardization, of establishing standards for comparison. This in turn has at least two main components: The first is the problem of pattern—what kind of pattern of human behavior are we going to be concerned with; and the other concerns the question of impairment—of the degree to which these patterns handicap the individual in the social medium of which he is a part.

A serious matter in this problem of standardizing our point of reference, is the question of environmental components that may be part of the diagnostic process itself, that is: the built-in environmental factors that are there because of the way diagnoses are made. Such can obviously lead to misconceptions of cause if not recognized.

Another related problem that has been touched upon today, is the

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one that comes up when we ram together the total collection of what is seen in a mental hospital and call it mental illness—schizophrenia, psychoneurosis, alcoholism, sociopathic types of behavior, etc. This is certainly an exceedingly heterogeneous group of human behaviors, and it makes one wonder whether he would generalize about admissions—first admissions or any other—to a general hospital as is commonly done about first admissions to a mental hopital. Yet, after all, is a general hospital any more heterogeneous in its collection of different kinds of things than a mental hospital?

A third problem is the question of what are noxious factors: What are to be considered as damaging factors in the origin, the course and the outcome of the phenomena we call psychiatric disorder? There is a need here to give thought to benign and neutral aspects. An otherwise damaging constellation of events can be rendered neutral or even benign because of particular factors which enter. Hysterectomy is an example: in one situation it is stressful; in another situation it may be psychologically benign.

A point which I think is pretty closely related to the previous one, is the need for an explicit statement of the psychological frame of reference in thinking about cause. This has been implicit but only implicit all day in many of the things that have been said. Various assumptions were made as to how psychological mechanisms work and yet the underlying framework that any one of us holds could be quite different from others in the group here. So it would seem to me desirable to have these assumptions made more articulate than they were. It might be wise to have clear concepts as to the framework of ideas as is the case in other forms of epidemiology with regard to infection, nutritional deficiencies, etc. I think there was some obscurity in our implicit references as to how something might be benign or noxious so far as the origin, course and outcome of psychiatric disorders are concerned.

The last point has to do with the emphasis that was given to the necessity for interplay between the epidemiologic approach and the clinical and laboratory approach. The separation of these is an unhappy situation. Allied to this is the business of interrelating extensive study with intensive study. While we can very often get one kind of reliability, if not validity, with an extensive study which deals in large numbers, we are apt to lack knowledge as to why we get these associations. The intensive small study that is geared to the ex-

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tensive study may provide at least something better than exists now.

Mr. Allen, the late editor of *Harper's,* used to tell a story which I think is apposite to the fix we are in here. He said he had a roommate at Harvard who didn't study very hard but lay around, apparently thinking. Along towards the third year Allen got out of this man what he was doing. He said he was spending his time trying to be famous, and that the quickest way was to have a law named after you: thus Newton was known for Newton's Law and so on. In his fourth year he hit upon it: "If you play with anything long enough, it will break."

That set Allen to work, and he established Allen's Law, which states that "Everything is more complicated than you think."

Before I turn in my gavel, I want to express my very deep appreciation to all of you for making the task of the chairman so easy today.

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